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Structural Changes Related to Texture of Pre-Peeled Potatoes

ABSTRACT

Pre-peeled potatoes, treated with heated ascorbic/citric acid solutions to control browning, undergo firming and separation of superficial tissues that affect texture after mashing and slicing following cooking. Examination of superficial parenchyma cells in cooked potatoes by scanning and transmission electron microscopy indicated greater cell wall rigidity and middle lamella retention in samples treated with browning inhibitors than in untreated controls. Lumps in mashed potatoes prepared from treated samples showed similar characteristics. Treated samples sometimes displayed a discontinuity beneath the superficial tissue, corresponding to site of tissue separation during slicing. These observations are consistent with treatment-induced changes in cell wall and/or middle lamella pectins as the cause of such textural defects.

Key Words: pre-peeled potato, browning inhibitors, texture, microscopy

INTRODUCTION

PRE-PEELED POTATOES require treatment with browning inhibitors to control discoloration during storage. Pre-treatment of peeled potatoes with heated ascorbic (AA) and citric acid (CA) solutions, prior to a browning inhibitor dip containing 4% AA, 1% CA and 1% sodium acid pyrophosphate (SAPP), has greatly extended storage life over that after the dip alone (Sapers and Miller, 1995). However, under some conditions, such pre-treatment induced changes in the cooked product that resulted in surface firming (case hardening) and partial separation of the case-hardened superficial layer from underlying tissue during slicing. Furthermore, case-hardened potatoes formed large lumps during mashing. Such defects would greatly limit the utilization of pre-peeled potatoes, which had received the anti-browning treatment. Case hardening has been reported as a result of steam peeling (Feinberg et al., 1987) and from use of commercial browning inhibitors (Garrett, 1995). Such firming may be related to pectin methylesterase (PME) activation induced by heating and subsequent cross-linking of demethylated pectin (Bartolome and Hoff, 1972). Also, acidification appears to firm sweet potato tissue by a chemical rather than an enzymatic process (Walter et al., 1992). Firming also may result from chilling-induced retrogradation of gelatinized starch (Reeve, 1970, 1972). However, at relatively low temperatures used in our browning inhibitor treatments, notable starch gelatinization would not be likely (Roberts and Proctor, 1955; Kubota et al., 1979; Pravisani et al., 1985).

Textural defects have been reduced but not eliminated by empirical modification of treatment conditions, i.e., omission of calcium from the browning inhibitor formulation, reduction in treatment temperature, and addition of EDTA to the heated AA/CA solution (Sapers and Miller, 1995). They still might represent a potential problem for users of the technology. A better understanding of the nature of such defects would help develop approaches to avoid them. Our objective was to determine the cellular and ultrastructural changes causing textural defects induced by browning inhibitor treatments.

MATERIALS & METHODS

Raw materials and processing

Norkotah and Russet Burbank potatoes, used for steam peeling trials, were obtained from commercial processors and stored for 1–4 wk at 20°C before processing. Generic Russet and round-white potatoes, used for abrasion peeling trials, were obtained from local distributors and stored for 1 wk or less at 20°C before processing.

Prior to peeling, potatoes were washed with water and sanitized by dipping in 100 ppm Cl_2 (sodium hypochlorite solution adjusted to pH 6.5 with citric acid) for 1 min, and rinsing briefly with water. Washed potatoes were peeled either by high pressure steam at 1400 kPa for 15.5 sec or by abrasion for 75 sec, as reported (Sapers and Miller, 1995). Following peeling, potatoes were briefly stored submerged in a solution containing 2% SAPP + 0.25% NaCl to provide temporary control of after-cooking darkening and enzymatic browning until all browning inhibitor treatments could be applied.

To control browning, peeled potatoes were heated in a solution containing 2% CA + 1% AA + 500 ppm EDTA for 5–8 min at 45–55°C, as reported (Sapers and Miller, 1995). The potatoes were cooled in running tap water at ≈18°C for 2 min and then dipped for 5 min in a 3% solution of Potato Fresh™ (PF; a commercial browning inhibitor, supplied by EPL Technologies, Inc. (EPL)). Treated potatoes were drained and impulse sealed within bags formed from Respire™ P369 (polyethylene-type film, oxygen transmission rate ≈4650 cc/m²/day supplied by EPL) to provide a reduced O_2 atmosphere during storage. Samples were given the heated browning inhibitor treatment followed by the PF dip, only the PF dip, or no treatment (controls) and were stored briefly at 3°C in the dark before preparation for microscopy.

Sample evaluation and preparation for microscopy

Treated and control pre-peeled potato samples were prepared for microscopy, either in the raw state or after cooking by boiling until tender (20–35 min, depending on raw material and treatment). A portion of the cooked samples was examined for case hardening. A wedge, cut from a cooked potato, was observed for separation of a tough superficial layer and resistance of that layer to mashing with a fork. In a more definitive study of mashed potato consistency, three raw tubers from each treatment were quartered and boiled in 2L water containing 3.4g NaCl for 20–30 min, with stirring every 3 min. After draining and cooling for 5–10 min, firmness measurements were made with a McCormick Fruit Tester (Model FT 011; McCormick Fruit Tech., Yakima, WA), using an 8 mm diameter plunger head. The potato quarters were transferred to a 4.73-L stainless steel mixing bowl and combined in an electric mixer (Kitchen Aid Model K5SS, Hobart Corp., Troy, OH) at speed setting 3, first with 22 mL butter for 30 sec, and then with 39.4 mL 1% lowfat milk for 4 min (Rombauer and Becker, 1974). At 2 and 3 min, the mixer was stopped so that the sides of the bowl could be scraped. A 100-g portion of each mashed product was suspended in 1L deionized water, stirred vigorously for 45 sec, and poured through a stainless steel No. 8 standard sieve (2.36 mm sieve opening, Newark Wire Cloth Co., Newark, NJ) to isolate larger lumps and unmashed tissue. The collected lumps were re-suspended in 1000 mL water and re-sieved twice to remove adhering small potato particles. The sieve was drained for ≈5 min, blotted dry, weighed, and the percentage of lumps not passing through the sieve was calculated. The lengths of the largest particles in each sample were estimated, and a selected portion was prepared for microscopic examination.

Comparisons of firmness and lumpiness were carried out with cooked Russet (Expt. R) and round-white (Expt. RW) potatoes given no treatment other than cooking (controls), potatoes dipped in PF, or potatoes heated in AA/CA/EDTA solution at 55°C, followed by dipping in PF. In Expt. RTC (Russet, temperature comparison), the 55°C treatment was compared with one at 45°C, with and without addition of 1% NaCl to the PF dip, using Russet potatoes. In the RTC experiment, we also measured the percentage of lumps in mashed samples as described.

Microscopic evaluation

A 4-mm thick slice was taken from the middle region of each whole potato with a stainless steel razor blade. One-quarter of the slice was immersed in 30 mL of 1% glutaraldehyde in 0.1M imidazole-HCl (pH 6.8) and stored at 4°C. A 4–5 mm deep trapezoidal slab was cut from the outside (peeled side) of the quarter slice, dehydrated in ethanol, and critical point dried. The specimen was mounted on a 25 mm diameter Al specimen stub with comparison slabs and coated with a thin layer of gold by DC sputtering. Lumps, isolated from mashed potatoes, were prepared for microscopy by the same procedure. Samples were examined in the secondary electron imaging mode with a JSM 840A scanning electron microscope (JEOL USA, Peabody, MA).

Samples for TEM were removed from 1% glutaraldehyde/0.1M imidazole HCl buffer washed in 0.1M imidazole buffer and fixed in 2% OsO_4. They were then washed in water, dehydrated in a graded ethanol series and embedded in an epoxy resin mixture. Finally, samples were thin sectioned with a microtome using a diamond knife, and stained with solutions of uranyl acetate and lead citrate. Specimens were examined in the bright field image mode with a Philips CM12 STEM.

RESULTS & DISCUSSION

Textural characteristics of treated potatoes

Cooked pre-peeled potatoes, that had been treated with heated AA/CA/EDTA solution for 5 min at 45–55°C and given a 5 min PF dip to control browning (identical to samples prepared for microscopy) were examined to confirm presence of textural defects reported previously (Sapers and Miller, 1995). High pressure steam-peeled Russet Burbank and Norkotah potatoes, that had been treated to control browning and cooked, showed slight toughening and separation of a surface layer about 2–3 mm thick. Treated and cooked, abrasion-peeled generic round-white potatoes were tender but showed more severe separation of the surface layer than did the high pressure steam-peeled samples. However, in other comparisons, we have not observed a consistent relationship between method of peeling and textural defects.

Cooked Russet potatoes that had been abrasion peeled and treated with heated AA/CA/EDTA solution, followed by the PF dip, were firmer (more case hardening) than untreated controls, based on penetrometer readings (Table 1). Mashed potatoes, prepared from treated samples were lumpy and contained larger particles than did mashed, untreated controls where consistency was smooth and lump size was <0.1 cm. Potato firmness but not lump size decreased when boiling time was increased (Expts. R and RW). Treatment with AA/CA/EDTA at a lower temperature resulted in decreased firmness, a smaller proportion of sievable lumps, and a reduction in maximum lump size (Expt. RTC). Addition of NaCl to the browning inhibitor dip decreased firmness of cooked potatoes, given the 55°C/5 min treatment, but did not reduce maximum lump size (Expt. RTC). Limited comparative data indicated that the proportion of sievable lumps did not correlate highly with the maximum lump size.

The case hardening appears to be similar to the firming effect reported previously in pre-heated potatoes (Bartolome and Hoff, 1972), carrots (Quinteros-Ramos et al., 1992), and other products. Such heat-induced firming must be distinguished from softening and cell sloughing, induced by cooking (Sterling and Bettelheim, 1955; Hughes et al. 1975a; Reeve, 1977). The decreased case hardening at the lower treatment temperature may have been due to reduced PME activation, resulting in less demethylation and subsequent cross-linking. Temperature reduction may provide effective means of reducing the severity of case hardening, but it also would decrease the effectiveness of the treatment in controlling browning (Sapers and Miller, 1995).

Table 1—Effect of browning inhibitor treatments and boiling time on potato firmness and mash lumpiness

Expt.	Treatment	Boiling time (min)	Penetrometer (kg)	Lumpiness (%)[e]	Lumpiness Max. size (cm)
R (Russet)	Untreated	20	0.62 ± 0.18	—	0.1
	PF[b]	20	0.78 ± 0.09	—	0.3–0.6
	55/5 + PF[c]	20	0.98 ± 0.21	—	0.5–1.0
	55/5 + PF[c]	30	0.53 ± 0.09	—	0.5–1.0
RW[a]	Untreated	21	0.42 ± 0.09	—	0.05
	PF[b]	21	0.62 ± 0.16	—	0.5
	55/5 + PF[c]	21	0.87 ± 0.32	—	0.5–1.0
	55/5 + PF[c]	30	0.53 ± 0.09	—	0.5–1.0
RTC (Russet)	55/5 + PF[c]	27	0.68 ± 0.08	18.8	0.25–1.0
	45/5 + PF[c]	27	0.48 ± 0.10	17.1	<0.5
	55/5 + PF + NaCl[d]	27	0.50 ± 0.03	22.6	>1.0
	45/5 + PF + NaCl[d]	27	0.44 ± 0.09	15.2	<0.5

[a] RW=round-white.
[b] Dipped in PF for 5 min.
[c] Heated in 1% AA + 2% CA + 500 ppm EDTA for 5 min at 45° or 55°C; then dipped in PF for 5 min.
[d] Same as b but 1% NaCl added to PF dip.
[e] Lumps not passing through a No. 8 standard sieve.

Firming also may have been induced by exposure to acids during treatment, as reported by Walter et al. (1992) with sweet potato. Hughes et al. (1975b) also reported a firming effect of acidification with white potato that could be overcome by extending cooking, as we found. Our data showed some reduction in firmness by addition of NaCl to the browning inhibitor dip (and ultimately to the cooking water), as predicted from their results. However, we could not consistently overcome treatment-induced lumpiness in the mashed product by this treatment. Apparently, the firming effects of browning inhibitor treatments (both heating and acidification) could be partially reversed by prolonged cooking and the presence of Na^+ in the cooked product. Addition of NaCl to precooked potatoes and other vegetables prior to dehydration greatly reduced their reconstitution times (Truckenbrodt and Sapers, 1970). Salt addition to snap beans resulted in enhanced softening during cooking which was attributed to Ca^{++} displacement by Na^+ and to greater pectin solubilization, perhaps due to β-eliminative depolymerization (Van Buren, 1983; 1984; 1986). In contrast to firming, lump formation appeared to be largely irreversible.

Examination of abrasion-peeled potatoes by SEM

The surface of specimens from raw, abrasion-peeled Russet potatoes, cut normal to the plane of the tuber surface (Fig. 1A), displayed a cross-sectional view of parenchyma cells underlying the peeled surface, with well-defined cut (sheared) cell walls, which appeared to be empty or contained varying numbers of starch granules. The presence of cut or broken cell walls in sheared potato tissue is characteristic of raw tissues. Broken cell walls were observed at the rupture surface of raw potato specimens subjected to shear strength determinations (Sterling and Bettelheim, 1955). Structural failure of raw potato flesh, subjected to compression or torsion, occurred by rupturing of cell walls rather than by cells breaking away from each other (Diehl and Hamann, 1979). Raw samples, treated with browning inhibitors at 55°C, were similar in appearance to controls and showed no evidence of starch gelatinization due to treatment at the elevated temperature (Fig. 1B).

Cooked, peeled controls (Fig. 1C) showed parenchyma cells filled with a solid mass and embedded in an ordered structure in which cell boundaries but not individual cell walls were observed. In contrast to raw controls, cell walls of cooked controls did not appear to be cut. Some detached or loosely attached parenchyma cells were seen against the ordered background. These observations were consistent with tissue rupturing at the middle lamella rather than across cell walls (Sterling and Bettelheim, 1955; Reeve, 1970; 1972). However, superficial tissue from cooked potatoes, treated with browning inhibitor solution

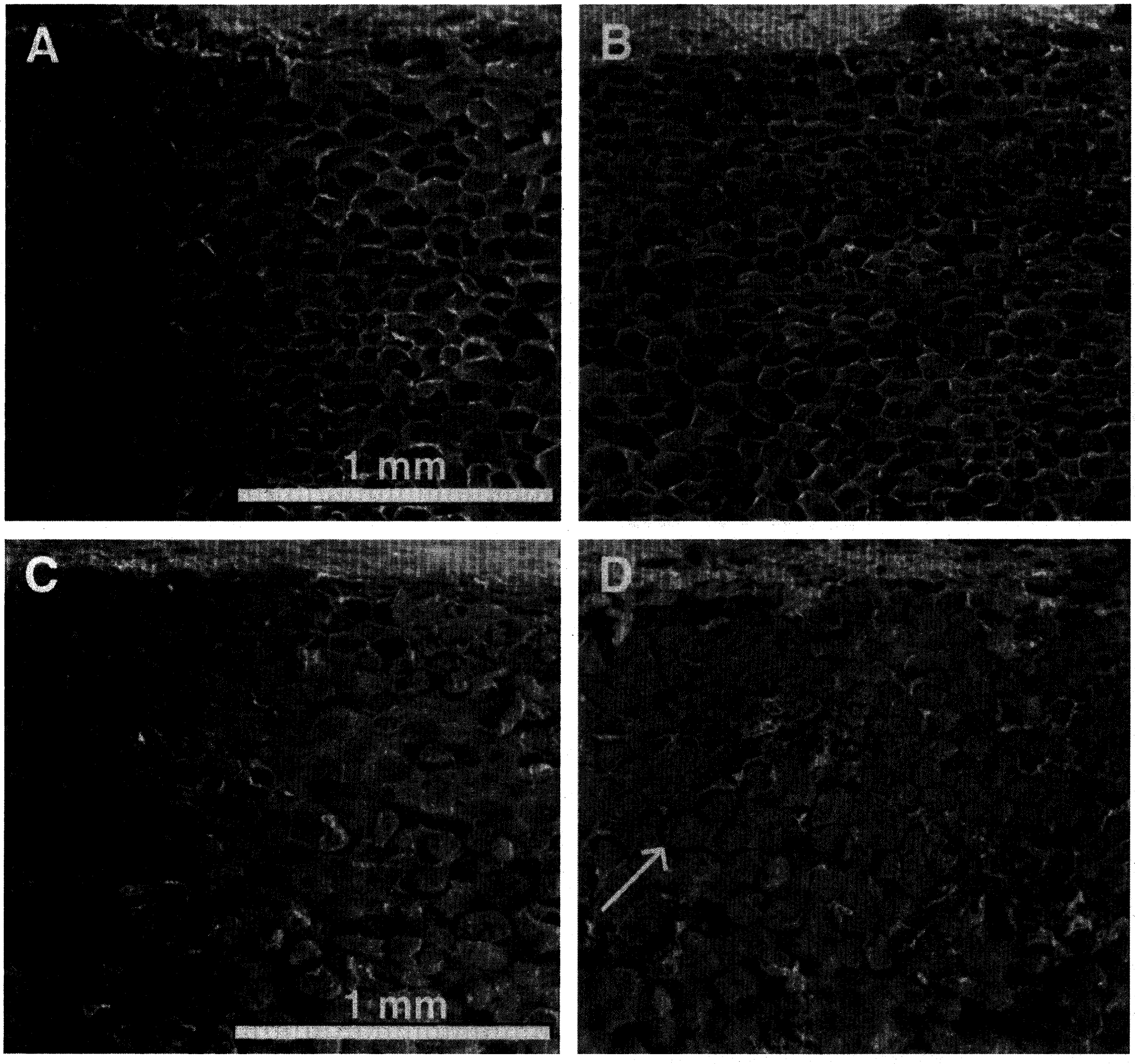

Fig. 1—SEM micrographs of abrasion-peeled Russet potatoes: (A) raw, control; (B) raw, treated with AA/CA at 55°C for 5 min followed by PF dip; (C) cooked control; (D) cooked, treated with AA/CA at 55°C for 5 min followed by PF dip. Bar represents 1 mm. Arrow (1D) indicates separation between cut cell walls and solidified cell contents.

at 55°C (Fig. 1D), showed numerous parenchyma cells, cut in cross section, in which the solid mass had shrunk or pulled away from rigid-appearing cut cell walls and where few detached cells were visible. These cut cells were in a layer about 1–2 mm thick, which overlaid parenchyma cells similar to those in cooked controls. Samples given the different heated browning inhibitor treatments were generally similar in structure. Likewise, treated round-white potatoes showed the same structural features as Russet potatoes (data not shown).

A similar but less extensive structural feature was observed in cell layers underlying the peeled surface of potatoes that had been dipped in Potato Fresh™, without prior treatment with heated browning inhibitors (Fig. 2). Apparently, exposure to the organic and inorganic acids in Potato Fresh™ and other commercial browning inhibitors induced some case-hardening, as reported by Garrett (1995).

Some changes in samples given the heated browning inhibitor treatments may have been due to heat alone rather than to the combined effects of heat and browning inhibitor chemicals. However, the occurrence of case hardening in potatoes treated with browning inhibitors with no heat and absence of case hardening in samples heated in water without inhibitors (Sapers and Miller, 1995) makes this unlikely. The mechanism by which browning inhibitors induce firming is not clear since pH changes resulting from the treatment would be transitory due to the buffering effect of the potato tissue. Walter et al. (1992) attributed a similar firming response to acidification in sweet potato tissue to attainment of a pH range (5.9 to 3.8) in which both β-elimination and acid hydrolysis had minimal effects, as postulated by Doesburg (1961). Alternatively, endogenous Ca^{++} and Mg^{++} might be solubilized and redistributed by browning inhibitor acids diffusing into treated potato tissue, resulting in greater cross-linking of cell wall pectins.

In some treated and cooked pre-peeled potatoes, a discontinuity or crack, corresponding to the region where a case-hardened outer layer would separate, was seen (Fig. 3). Cells on either side of the discontinuity were similar in appearance. A boundary was observed between altered parenchyma cells having rigid-appearing cut cell walls and more normal appearing parenchyma cells. However, the discontinuity did not occur at that point but at least 1 mm beneath the boundary.

Examination of high pressure steam-peeled potatoes by SEM

Raw, steam-peeled Russet Burbank potatoes, either untreated or treated with heated browning inhibitor solution, showed an outer layer of parenchyma cells, about 1 mm thick. In that layer

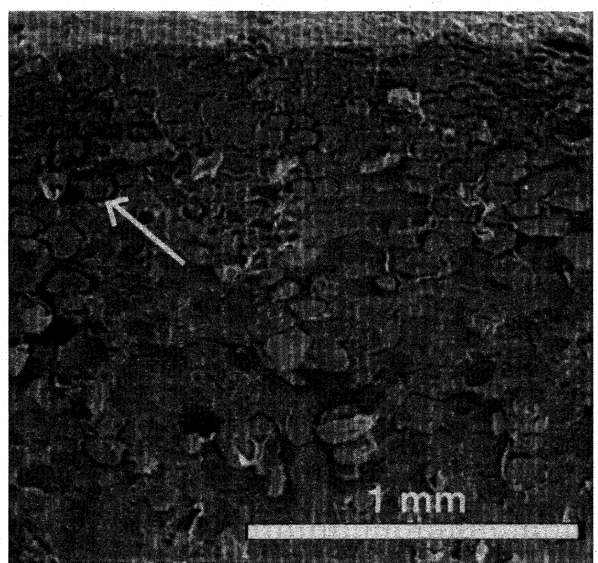

Fig. 2—SEM micrograph of cooked, abrasion-peeled Russet potatoes treated with PF dip. Bar represents 1 mm. Arrow indicates separation between cut cell walls and solidified cell contents.

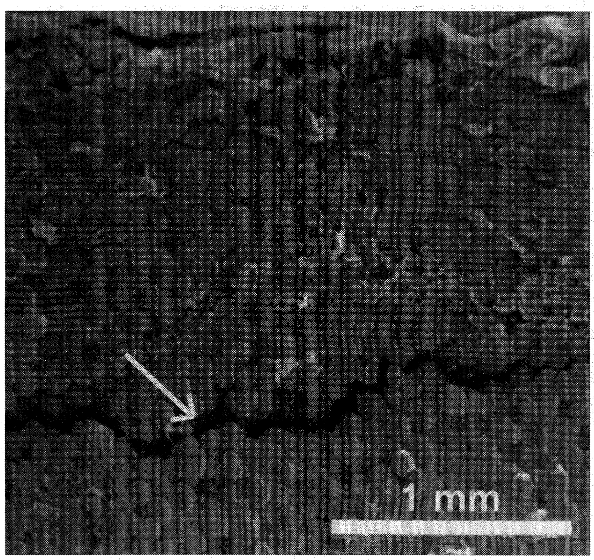

Fig. 3—SEM micrograph of cooked, abrasion-peeled round-white potatoes treated with AA/CA at 55°C for 5 min, followed by PF dip, showing discontinuity (at arrow). Bar represents 1 mm.

most cells contained a shrunken solid mass, presumably gelatinized starch, within distinct and rigid appearing cell walls. These were similar in appearance to superficial parenchyma cells of treated and cooked abrasion-peeled potatoes (Fig. 4A). The shrunken mass was separated from cell walls by a gap of several μm. The gelatinized layer corresponded to the 1–1.5 mm thick heat ring, visible in peeled raw potatoes, that has resulted from surface cooking by steam (Smith and Huxsoll, 1987). This heat ring was usually not as thick as the layer of separated tissue, seen in cooked, treated potatoes, which was about 2–3 mm thick. Beneath the heat-ring layer, parenchyma cells of treated potatoes were empty or contained clusters of starch granules within well-defined, cut cell walls. There was no indication of starch gelatinization, similar to the appearance of raw, unpeeled specimens.

With treated and cooked, steam-peeled potatoes, cells within the heat ring were largely unchanged by cooking, showing shrunken gelatinized masses enclosed by cut cell walls (Fig. 4B). Whether browning inhibitor treatments significantly increased the thickness of this layer could not be established. Beneath the heat ring, parenchyma cell walls were not cut, and cells were similar in appearance to untreated, cooked potato.

Changes in cell walls induced by treatment

Thin sections of treated and control pre-peeled potatoes were examined by TEM for changes in cell wall structure that might correlate with case hardening and formation of cracks (discontinuities). Cell walls of cooked, abrasion-peeled Russet potatoes, treated with heated browning inhibitors (Fig. 5A), appeared more electron dense, with greater retention of the middle lamella than cell walls of cooked controls (Fig. 5B). In contrast, treatment of round-white potatoes (Fig. 5C) increased retention of electron dense cell wall material near cell wall boundaries over that in cooked controls (Fig. 5D), but there were no visible effects on the middle lamella. Van Marle et al. (1997) reported depletion of electron dense material in cell walls of tissue from potato disks that had been boiled for 15 min. Cell walls of uncooked potato tissue and tissue boiled for only 5 min resembled cell walls from treated potatoes in our study in retention of electron dense material and apparent rigidity.

Cell walls located immediately above the discontinuity in cooked, treated steam-peeled Russet Burbank potatoes (Fig. 6A) contained more electron dense material than did cell walls below (Fig. 6B), although cells from both regions appeared normal by SEM. This suggests that case-hardening effects, induced by browning inhibitor treatment, extended beneath cell layers showing cut and rigid-appearing cell walls. Separation may occur at a depth below these cell layers where the strength of the cell wall-middle lamella complex falls below some critical level. At that depth, cell wall rupturing along the middle lamella may occur as a result of thermal expansion during cooking. A similar hypotheses was proposed to explain cracking during boiling of potato slices, or from compressive and shearing forces produced by slicing (Reeve, 1977). Starch gelatinization within the heat ring may have contributed to case hardening and separation in steam-peeled potatoes but would not explain the case hardening and separation seen in abrasion-peeled potatoes. We have seen no indications that stresses or tissue damage caused by abrasion peeling contributed to separation, and such separation did not occur in cooked abrasion-peeled controls.

Appearance of lumps by SEM and TEM

Comparison of small particles (through No. 8 standard sieve) and larger lumps from mashed, treated potatoes by SEM showed the small particles (associated with smooth mashed potato consistency of controls) were individual parenchyma cells or small clusters of cells (Fig. 7A). The large lumps (a major defect in mashed, treated potatoes) were multicellular structures, probably derived from separated superficial tissues (7B). Individual cells within lumps varied greatly in appearance but showed some characteristics of raw potatoes. They had rigid-appearing cut cell walls with cellular contents either missing or remaining within cell walls but with some apparent shrinkage, as found in cooked treated samples.

Comparisons of small particles and lumps by TEM showed considerable variation in cell wall structure, as expected for cells derived from different tissues within the intact potato. Nevertheless, lumps tended to retain more electron dense material in the cell wall/middle lamella structure (Fig. 8A) than did small particles (Fig. 8B). Presumably, large lumps originated as case-hardened superficial tissue and thus would show greater middle lamella retention than small clusters of cells originating beneath this tissue.

We hypothesize that the structural changes associated with case hardening and lump formation were initiated by heating. Within the temperature range used for browning inhibitor treatment, this could activate PME, resulting in partial demethylation of pectin and the creation of new binding sites for endogenous

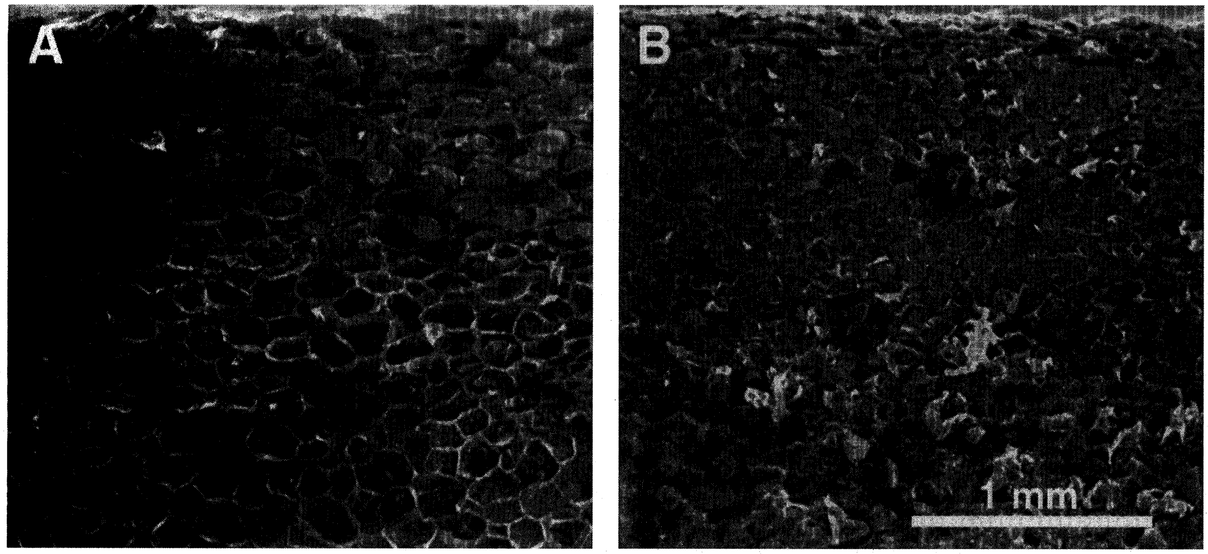

Fig. 4—SEM micrographs of steam-peeled Russet Burbank potatoes treated with AA/CA at 55°C for 8 min, followed by PF dip: (A) raw; (B) cooked. Bar represents 1 mm.

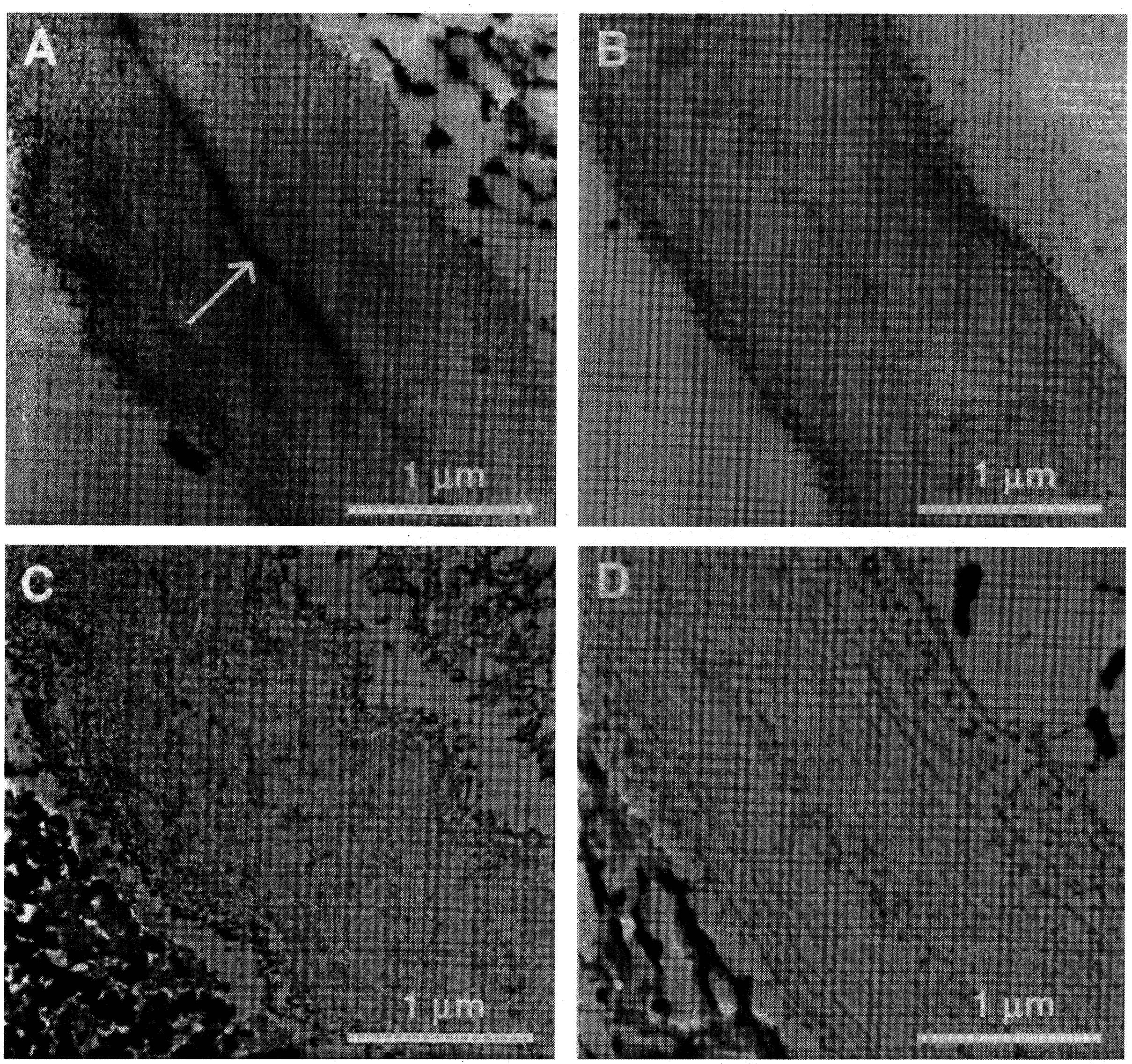

Fig. 5—TEM micrographs of cell walls of cooked, abrasion-peeled potatoes: (A) Russet, treated with AA/CA at 55°C for 5 min, followed by PF dip, showing middle lamella retention (arrow); (B) Russet untreated control; (C) round-white, treated with AA/CA at 55°C for 5 min, followed by PF dip; (D) round-white untreated control. Bar represents 1 μm.

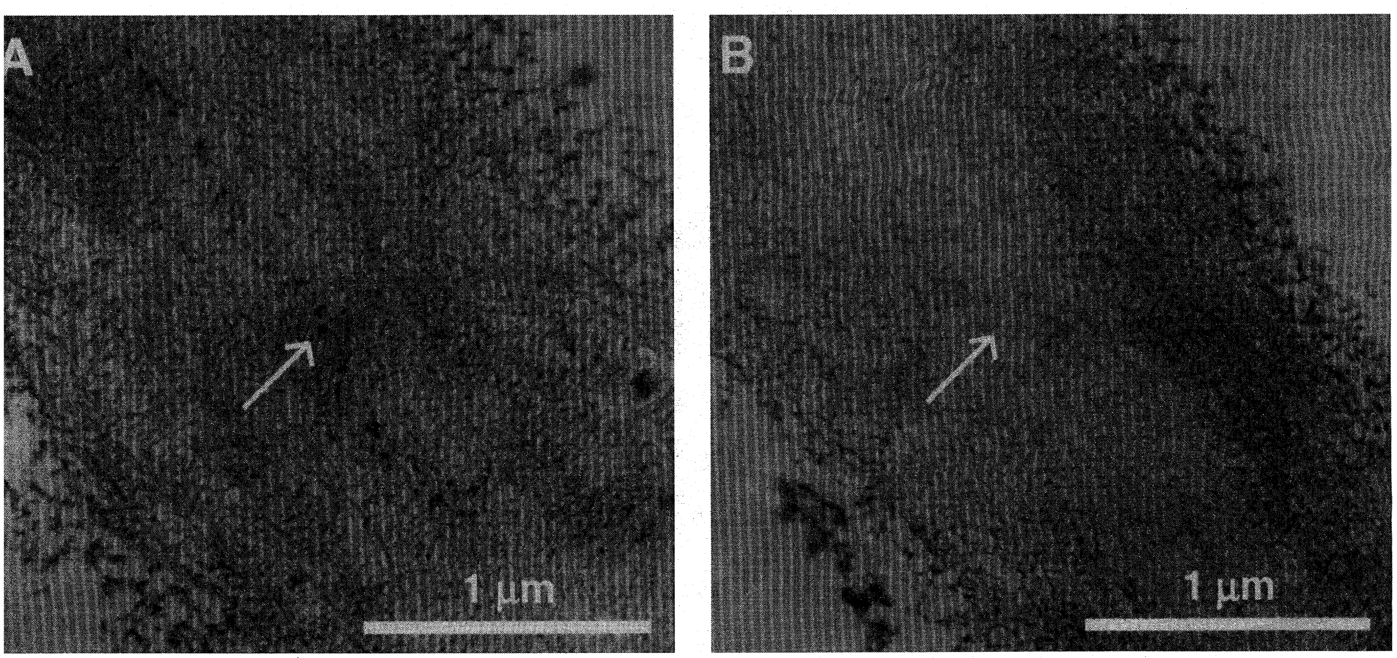

Fig. 6—TEM micrographs of cell walls of cooked, steam-peeled Russet Burbank potatoes, treated with AA/CA at 55°C for 8 min, followed by PF dip: (A) immediately above discontinuity; (B) below discontinuity. Bar represents 1 μm. Arrows indicate variation in electron dense material in vicinity of middle lamella.

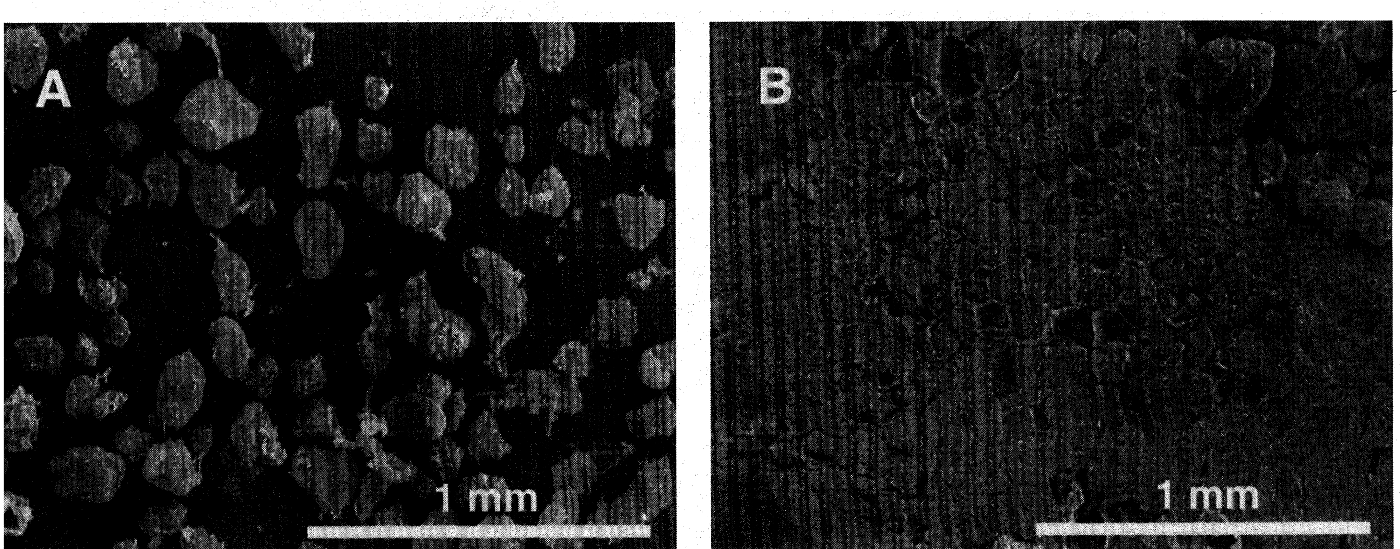

Fig. 7—SEM micrographs of small particles and lumps from mashed cooked Russet potatoes, treated with AA/CA at 55°C for 5 min, followed by PF dip: (A) small particles; (B) lumps. Bar represents 1 mm.

calcium or magnesium (Bartolome and Hoff, 1972). Diffusion of acids into the affected tissue could mobilize and redistribute calcium (or magnesium) from cell interiors to cell walls so that cross-linking could occur, possibly when tissue pH had equilibrated at a higher value as a consequence of buffering. Previously, we observed an elevation in surface pH from $\approx$3 immediately following treatment to $>$4 within 24 hr (Sapers and Miller, 1992). Cross-linking would result in greater retention of pectins (presumed to be the electron dense substances seen by TEM) in the cell wall-middle lamella complex during cooking. Thus, walls of parenchyma cells near the peeled surface would be stronger in treated samples than in controls and would appear more rigid in cross sections by SEM. The depth below the peeled surface to which such processes extend would be governed by the extent of heat penetration and consequent PME activation. This would be determined by treatment time and temperature and post-treatment cooling conditions, and by the extent of acid diffusion during treatment. Below that depth, the tissue would be largely unaffected by treatment. When treated potatoes were cooked, the parenchymal tissue in which cell walls were strengthened by cross-linking would hold together better than underlying tissue and would separate from it when subjected to thermal expansion or slicing. The site of separation, $>$1 mm below the boundary between altered and normal-appearing parenchyma cells, suggests the presence of a strengthened cellular structure extending many cell layers below the boundary. The decrease in firmness after prolonged cooking and salt addition may have been due to partial disruption of the cross-linking process. However, such treatments did not prevent case hardening and consequent formation of large lumps during mashing or weaken lump structure so that lump size would be reduced substantially during mashing.

The apparent increase in cell wall rigidity (tissue failure by cell wall rupturing rather than by separation along the middle

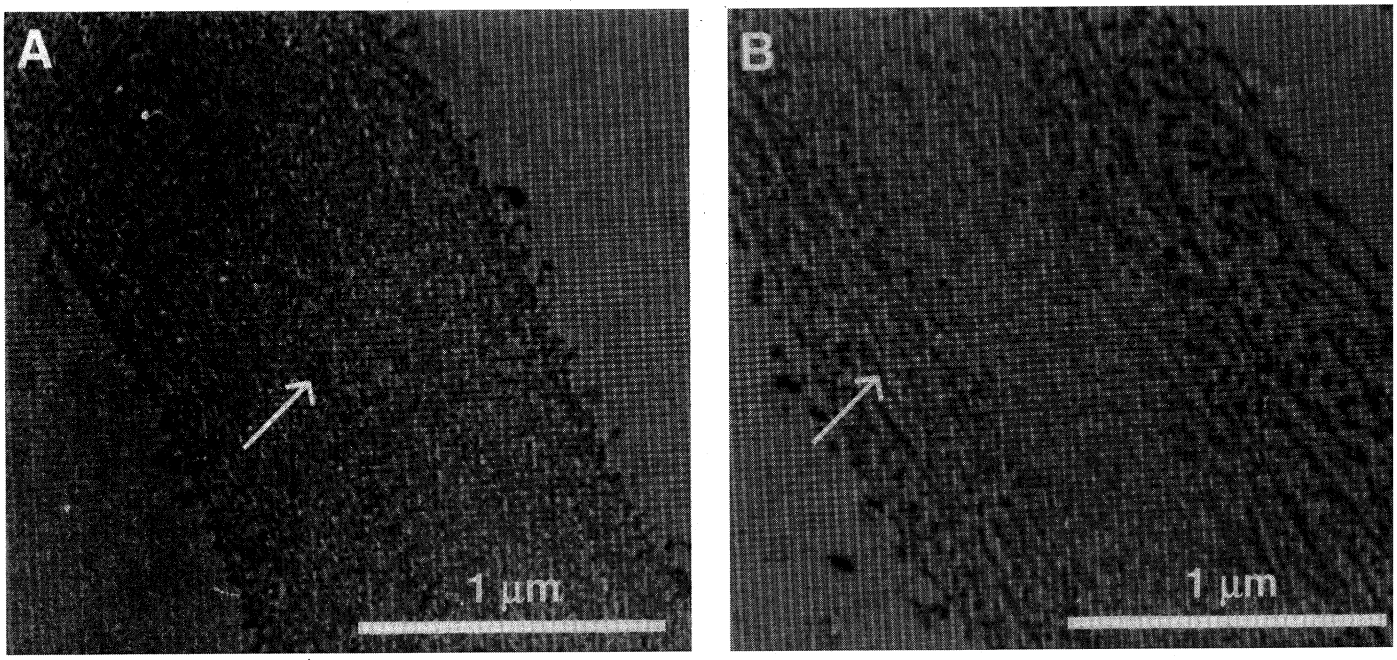

Fig. 8—TEM micrographs of cell wall structure in small particles and lumps from mashed cooked round-white potatoes, treated with AA/CA at 55°C for 5 min followed by PF dip: (A) lump; (B) small particles. Bar represents 1 μm. Arrows indicate variation in electron dense material in vicinity of middle lamella (8A) and at the cell wall boundary (8B).

lamella) was accompanied by increased retention of electron dense cell wall material in cooked, superficial potato tissue, induced by browning inhibitor treatment. These were due to chemical alterations of the cell wall/middle lamella pectins that resulted in increased pectin retention during cooking and cell wall strengthening (Bettelheim and Sterling, 1955). On a macroscopic scale, such structural changes would produce the case-hardening observed in cooked samples.

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